I have purposely avoided mentioning the other changes occurring in the eye and other portions of the system from tobacco and alcohol, limiting my paper to the chronic retro-bulbar neuritis. Other drugs capable of producing similar changes to the above are strammonium, cannabis indica, chloroform, chloral, opium, bi-sulphide of carbon, nitrobenzol, arsenic, lead, iodoform, and the toxin of diabetes.

A CASE OF PYLORIC STENOSIS IN THE NEWLY BORN.*

By HENRY J. KREUTZMANN, M. D. San Francisco.

On Sunday, the 10th of May, 1908, at 9:40 a. m., a baby boy was born to Mrs. A. E. in this city. Incidentally I might mention that about two years ago I had performed Alexander's operation upon the lady for retroversion and slight descensus of the uterus. Pregnancy (it was the first) was without mishap; the lady is of short stature, but her pelvic measurements being normal, delivery occurred without any difficulty in shorter time than usual, owing to a strong, muscular physique of parturient. The baby was perfectly normal; its weight at birth was 7¼ pounds, it acted in every way as a perfectly normal baby; it took the breasts and showed a phenomenal development, gaining 7 ounces the first week, 10 ounces the second week, and eleven ounces the third week after birth. On the eleventh day of its life I performed circumcision, the urethral opening in the prepuce being rather small.

When two weeks old the baby vomited for the first time. This vomiting, coming shortly after circumcision, was attributed to the disturbance brought about by the little operation. The next day the baby vomited again, once a day, then twice a day, then oftener. The vomiting occurred at first after the same meal, at 6 a. m.; this same 6 a. m. vomiting was kept up all along; to it was added another and another; finally the baby vomited also in the night time, which before he had not done; before he had kept all his night meals well. The vomiting took place soon after nursing; the quantity varied, also the manner, being sometimes the usual throwing up, at other times more forceful. There was considerable gas belched and passed per rectum. The passages were frequent but very good in consistency and color. The baby was sleeping, resting, gaining; for this reason no weight was attached to his vomiting for some time.

When the baby was just three weeks old, I saw for the first time the baby vomiting; it was soon after a meal; he had taken the breast very energetically and when he vomited, milk, partly coagulated, was expelled with a great deal of force, passing even through the nose. I was astonished and somewhat perturbed over this sort of a vomitus; but the baby appearing without any distress, I consoled myself with the old adage: "Babies who throw up —grow up," or I rather had in my mind the German word, Speikinder—Gedeihkinder, which means the same.

Under the circumstances, the baby thriving, sleeping and gaining, I did not oppose when the family wished to go to their summer home in Menlo Park. The trip was made in an automobile the next day, Monday, the baby sleeping on the way. After the arrival in the country the baby vomited more and began to show some restlessness; so Dr. Harry B. Reynolds of Palo Alto, to whom I had referred the family, was called. When the patient did not sufficiently improve under his treatment with change in diet, physics, lavage of the stomach and rectal feeding, I was called and I saw the baby on Tuesday, June 10th, in the forenoon, six days after the first visit by Dr. Reynolds.

At our first consultation the question of pyloric stenosis was not taken up. The baby was fairly well nourished yet; the inspection of the child failed to show anything peculiar; we looked upon the case as one of disturbed digestion, hard to explain though in its etiology. When after thirty-six hours of absolute inanition and continued vomiting, the baby was seen again, we both agreed that the case was one of stenosis of the pylorus. I felt distinctly the tumor in the region of the pylorus; but even then the distention of the stomach was not great, probably because nourishment was withheld at the time before examination.

To be prepared for every emergency the baby was removed to the Adler Sanitarium in this city the next morning, June 12th, 1908. Dr. Langley Porter was called in consultation, and from that time on we treated the patient conjointly.

During our observation the case presented a typical picture of pyloric stenosis in a baby, as characteristic as ever one has been described. Temperature kept normal, pulse at times rather rapid and weak. Patient vomited everything that he had occasion to. No fecal matter was discharged per rectum, only the residue of rectal feedings, mixed with bile stained secretion of the intestines. The urine became very scanty, concentrated, the salts of uric staining the diapers blood red. The supra-umbilical region was greatly distended, bulging; the distention was due to the stomach; at times the antiperistaltic waves of the stomach could beautifully be seen. The infra-umbilical region appeared insignificant. At times, not always, a button-like hardness could be felt in the region of the pylorus.

Our next effort was to keep the baby alive. He was kept warm, handled as little as possible; olive oil, later sweet lard, was rubbed into his skin, and alimentation carried on through his rectum. Besides this general idea of preserving his vital forces as much as possible, we resorted to three distinct attempts of therapeutic measures, calculated to overcome the pyloric stenosis.

1. Acting on the idea that the contents of the stomach, either gastric secretions or ingested food, was producing the obstacle through irritation, the stomach was washed, weak predigested whey, or Vichy water was given; no result.

2. The patient was at times very restless, appar-

*Read before the San Francisco County Medical Society.

ently in great pain, did not sleep; in order to relieve the suffering, to induce sleep and at the same time, possibly, to overcome the spasms that might cause the stenosis, opium, chloralhydrate, bromides were given; the result as far as pain and sleep were concerned, was obtained, but as far as the stenosis was concerned, the result was nil.

3. As a directly curative measure, regular teaspoon doses of olive oil were given per os upon the recommendation of some authority; no result.

The patient was losing weight and ground slowly but steadily. The critical time for the decision for surgical interference arrived. This could not so easily be determined. On one side we had to consider that an intra-abdominal operation in so small a baby constitutes a most serious undertaking; on the other side, the proper time for a possibly life-saving operation must not be lost; we did not wish to resort to the operation in extremis, with no chance for recovery. So after eight days of expectant treatment we decided for an operation. Doctor Cheney and Doctor Lewitt saw the patient and were kind enough to corroborate our diagnosis as well as our proposition for immediate operation.

Having knowledge that Dr. Stillman, shortly before, had successfully operated in a similar case, we suggested that he should do the operation in order to give the baby the very best chances.

On June 19th, 1908, posterior gastro-jejunostomy was performed by Dr. Stillman. The mass was seen and palpated but no time lost to find out much about it. The baby stood everything very well, was slightly collapsed, but responded to stimulation, soon took water and kept it down. For a few days he would occasionally vomit bile-stained mucus and we feared that the much dreaded circulus vitiosus might have been established—but everything went well; vomiting ceased, the bowels moved, the wound healed well—in one word he survived. It required a good deal of experimenting but finally a proper food was found and he soon began to show increase in weight, not rapid, not regular, but increase anyhow, and his weight is now 13 pounds. The abdomen shows the normal aspect of a healthy infant; digestion is perfect.

DR. WM. FITCH CHENEY.

I have seen four cases of pyloric stenosis in infants, all of them in consultation.

Case 1. Seen with Dr. R. W. Baum in February, March and April, 1905. This baby was born in January, 1905, nursed exclusively at the breast and remained perfectly well until three weeks' old. Then he began to vomit. The food was changed to a condensed milk mixture, then to peptonized milk, then nothing was given but a weak brandy solution; but vomiting persisted with all. His weight fell from 9 to 6⅞ pounds. I saw him first on February 28th, when he was seven weeks old. He was then greatly emaciated and had a very definite peristaltic wave across his stomach whenever the viscus was filled. Lavage always showed retention in the stomach. His weight had fallen to six pounds by early April. It then remained stationary for a time and then gradually the baby gained and vomiting ceased. By May 7th he weighed again 7⅝ pounds and after that gained continuously and normally. Both Dr. Baum and myself felt that peptonized milk in small amounts and daily lavage were what kept him alive.

Case 2. Seen with Dr. B. W. Stark on March 4th, 1908. The baby was then three weeks old, nursed at the breast from birth. He began to vomit at two weeks, apparently all food taken, shortly after nursing. This vomiting persisted in spite of milk sugar solution, condensed milk and peptonized milk mixtures. The food would frequently be retained for several feedings and then be vomited in large quantities. Emaciation rapidly occurred. Physical examination showed a definite peristaltic wave over the stomach and a palpable mass in the right hypochondrium, the size of the thumb. Lavage always showed food residue in the stomach. A gastroenterostomy was performed by Dr. Stanley Stillman on March 10th and the infant recovered.

Case 3. This was seen on June 9th, 1908, with Drs. Kreutzmann, Lewitt and Porter and has been described in detail by Dr. Kreutzmann. This infant was normal at birth, nursed at the breast, did well for three weeks and then vomiting began, persisting in spite of various kinds of food, with rapid loss in weight. The abdomen showed a peristaltic wave and a mass at the hypertrophied pylorus. Gastroenterostomy by Dr. Stillman on June 19th resulted in recovery.

Case 4. Seen in consultation with Dr. W. B. Lewitt, October 8th, 1908. This baby had been nursed at the breast exclusively but began to vomit on the 17th day of life and had vomited persistently since then, at first infrequently, gradually more constantly. After two or three nursings a large quantity would be vomited. There had been loss of weight from 8 pounds at birth to one ounce less than 7 pounds. The abdomen showed a peristaltic wave and a palpable mass as the pylorus. This case is still under medical treatment. Dr. Lewitt will report it in detail.

DR. LANGLEY PORTER.

It has been written, "We are most ignorant of what we are most assured." The worth of this axiom is never more clearly brought out than in its relation to the subject under discussion to-night. When one has seen a few little sufferers from congenital pyloric stenosis, one wonders that the condition escaped notice, until within recent times. It is true that Beardsley, of New Haven, in 1781 described a case with post-mortem notes, which seems to belong in this classification. And there are several other reports, notably that of Williamson (1841). It was not, however, until Hirshsprung, in 1887, reported three cases with autopsies that attention was centered on the condition and that observation and study were directed to it. Even at that time interest was aroused in but a few clinicians, chiefly Englishmen and Germans. Before 1808, while recorded cases were few and pathological data scanty, a school of observers, ably led by

Pfaundler of Munich, denied this symptom complex a place as a clinical entity; but in the face of accumulating material carefully reported from America as well as Europe, this position became untenable and these observers admitted the occasional occurrence of such a condition. But they doubted the diagnosis of a large proportion of the published cases, which proportion they assumed to be due not to a cognenital malformation but to a physiological error which had given rise to pyloric spasm. The Edinburgh and London men at this time were for the most part impressed with the idea that the condition was a true stenosis due to an obstructing pyloric tumor which was visible and palpable in the post-mortem room. As the matter stands to-day no one denies that cases occur during early infancy in which there is definite hyperplasia of the circular pyloric muscle with more or less complete stenosis, which cases present no signs or symptoms other than those referable to obstructions of varying degrees at the pylorus. The most characteristic of these signs are propulsive, cumulative vomiting, visible peristalsis of a large thickened stomach, shrunken hypogastrium, wasting, scanty urine and scanty meconium-like stools. The usual time of onset of these signs is in the latter weeks of the first month, most often in the third, as distinguished from the onset of symptoms immediately after birth in that very much rarer and always fatal condition, pyloroduodenal atresia. Why the onset of symptoms in hypertrophic stenosis is delayed no one has explained. Miller and Wilcox, however, have shown that vernin secretion is very much increased in coagulating power. We know that digestion is a developing function; we know that during the colostrum period vernin is not secreted by the human infant's stomach, and it may very well be that the appearance of this secretion is delayed in these cases, and that while there is no vernin, the milk, uncoagulated, can flow through a very narrow pylorus into the duodenum and there undergo digestion. But when late fermentation appears when coagulation takes place in the stomach, the physiological guardian action of the pyloric ring muscle becomes developed, and in its hypertrophied state it so overdoes its duty that the normally developed antagonistic longitudinal muscle cannot overcome its contraction, and obstruction is set up. I hope at a later meeting to report on the development of the vernin function of young babies.

There is a great diversity of opinion among the best minds of our profession in regard both to the etiology and to the treatment of the condition; the two extremes are represented by Hutchinson, Heubner and Bloch on the one hand, and Scudder and Morse on the other. The first hold with Pfaundler and attribute the obstruction to spasm of the stenosed pyloric ring muscle rather than to its hyperplasia alone. Hutchinson would go so far as to exclude all cases from operation, while Scudder and his colleagues consider that properly diagnosed cases of pyloric stenosis should at once be operated upon, and they attribute little value to spasm as a factor in the obstruction that occurs in such cases. The third school, which seems to me to have right with it, is led by Cautley, who since 1898 has seen more than twenty cases and who has given the matter very deep and earnest consideration. He admits the condition called by the Munich men, pure pyloric spasm, exists and thinks that many cases diagnosed pyloric stenosis that have spontaneously recovered are examples of this state. He further divides the cases of pure pyloric hyperplasia into those in which the circular muscle is so hypertrophied, the pyloric passage so narrow and the mucous membrane so folded that the stenosis is absolute and anatomical. The second class of case from his point of view is that in which although the hypertrophy is well marked, the pyloric canal is narrowed but to a slight degree, and in which the folds of the mucous membrane do not cause complete, continuous obstruction. Obstruction in this class of case he thinks may be attributed to irritative spasm of the hypertrophied pyloric sphincter, and patients suffering with this type of case under wise feeding may recover without other treatment. The first class in which I think are included all the cases reported to-night can be relieved only by operation and these patients will certainly die unless they are so relieved.

John Lovett Morse has recently published a most illuminating report on a case of this class which was successfully operated. Gastroenterostomy was done in the second month. During the eighth month the child died of a peritonitis, probably pneumococcic. After operation the child had developed in an absolutely normal way. On examination of the pylorus a perfectly characteristic tumor was found, spindle-shaped, obliterating the canal and extending into the duodenum, in appearance like the cervix uteri. In fact, the post-mortem findings differed not at all from the post-mortem findings obtained from other children, in whom death had been directly due to the obstruction of such a tumor. This case proves that at least some of these tumors do not alter as the Pfaundler-Bloch school teaches, and become patent in the later months of life. And such post-mortem findings clearly show that all cases of this disease are not due in any degree to spasm. As a student I saw a number of Cautley's cases and those of Voelecker and Newton Pitt and I have had the good fortune through the courtesy of Doctors Kreutzmann and Krone to be allowed to aid in the diagnosis and conduct of two cases in this vicinity, and Dr. Lartigau allowed me to be present at the operation of his patient; so in all, I have seen in eighteen months, three undoubted cases of this symptom complex of vomiting, pyloric obstruction, visible peristalsis, and depressed hypogastrium with scanty stools. I have further seen within the last few years four cases of intractable vomiting that correspond in type with the so-called pyloric spasm of the Pfaundler school as emphasized by Kopik in his recent paper. Personally I can see no justification for bringing these two groups of cases into juxtaposition. The spasm cases show a vomiting of vastly different type and do not have so marked hypogastric recession. The visible peris-

talsis in them is one that is so slight that it would escape attention were it not diligently searched for and since I have been looking for visible peristalsis of minor degrees, I have seen it in a number of little babies who, so far as one can judge, were suffering from nothing except too large amounts of food, or food of too high a fat content. Moreover, these babies are almost always bottle babies in contra-distinction to the stenotic babies who are usually breast fed. There seems to be no difference between this so-called pure pyloric spasm and the condition that arises in older infants accompanied by hyperchlorhydria.

In considering this morbid entity many interesting questions arise. A certain number of cases in which the clinical picture has been correctly drawn by excellent observers and which must have been true types of this disease, have gone on to recovery without operation. The validity of these observations cannot be doubted and the first question that arises is, "Why should we operate at all?" The answer to that is contained in the experience of untreated cases 89% of which have died, contrasted with the operative cases, 43½% have died. To quote the operative figures is not to make a fair statement of the cases either, because for a number of years after Hirschsprung's paper, diagnoses were delayed and operative interference was also delayed even after diagnosis, while the patient was treated expectantly. Moreover, even when the patient came to operation, operative procedures were more or less experimental. Pylorectomy, for instance, has been advocated, attempted and abandoned. Loretta's operation has also been practically abandoned in this country, Germany and England, although it is still the operation of choice in Scotland. Gastroenterostomy is only now being generally adopted, not because it is ideal but because it is practical, and the results, as you can realize from the cases reported to-night, are increasingly favorable. I have no doubt that in the near future when medical men are alert for this condition and bring their little patients early and in fair condition that the surgeon will save at least 90% of them.

When to operate is the second question that presents itself. Robert Hutchison, of London, is the only authority with extensive experience who unqualifiedly opposes operation under any circumstances. He justifies himself by the report of fourteen cases with one fatality. To quote him, including one case that died, the mortality has been one in fourteen. All the others have got well, and when I say well I mean perfectly well. I have followed these cases for three or four years and they remained perfectly healthy children, and I would say that the majority of cases, so far as I have seen, are not left with dilatation of the stomach; the condition seems to be one of genuine cure. Hutchison rejects the use of drugs, opium and belladonna, and where he has tried it, he rejects rectal feeding because he has found it impossible to make children retain for any length of time solutions given by rectum. He stakes his whole therapeutic attack in systematic diurnal stomach washing and in frequently feeding the child, if possible, small amounts of human milk, and if this is not feasible he gives thoroughly peptonized milk with an equal quantity of water. He calls attention to the danger of opium even in minute doses and cites one instance where 1-20 of a minim of the tincture given before each feeding for six or seven doses rendered the child comatose. He says in his experience relief cannot be expected for a long time. Although the children cease vomiting they go on losing weight for two or three months, and suddenly when the clinical picture seems blackest, they round the corner and begin to improve. His idea is that the disease is nothing else than a spasm of the hypertrophied pylorus and when the child is weak enough the spasm spontaneously gives way. He goes so far as to predict in the future operation will have no place in the treatment of this condition. So far at Hutchison's cases are concerned, I heard him report them in person, and in the discussion Cautley stated that he doubted very much the accuracy of the diagnosis; but allowing him full weight for the cases reported, considering the brilliant results that surgeons have achieved in this field, can we be justified if we allow these children to lag along in suffering and distress for four or five months and go on to a condition that will make a chance of successful surgical intervention slight, should that intervention become necessary? Personally, I believe not. I believe that stomach washing and medical treatment should be limited at the most to ten days or two weeks. If in that time there is no amelioration a surgeon should be called; but if during that space there is some improvement or even if the child but holds its own, then, unquestionably we are not justified in advising operation.

But after all is said and done, of this condition we know nothing at all save that we are very sure that some nurslings in their first month present a symptom complex that can be due only to obstruction at the pylorus and that some infants with such symptoms will have an enormously overgrown, hard, contracted pyloric sphincter. Beyond this we lose ourselves amidst the fogs of conjecture. We are not even certain that we have not included two or more conditions under one heading. Of the etiology we know nothing whatever. We are equally at a loss to explain the manner of onset even as we are to understand why some of the cases die while others go on to spontaneous recovery. We are indeed in this matter "most ignorant of what we are most assured."

Discussion.

Dr. Cheney: I simply wish to make a résumé of some of the points. Our object always with these babies is to save life and the essentials to saving life are accurate diagnoses and consequent careful consideration as to the means at our disposal for cure. In regard to the diagnosis, I wish to point out that there are two distinct classes of cases. There is hypertrophic stenosis where there is distinct organic thickening of the tissue and also that group of cases known as pyloric spasm which are possible to cure without surgical means. Our duty is to decide with which condition we are dealing. This matter has been gone over very carefully to-night. It is possible to form an opinion as to whether the case is

one of spasm or stenosis by the condition of the abdomen, by the difference in the peristaltic wave, the absence or faint rumbling of gas in the cases of spasm, or a palpable tumor in the stenosis which is in many cases present. It is not possible at a given time to decide this question and therefore the wisdom in delaying until we have watched the case in an effort to decide. By careful watching it is possible to decide whether we have stenosis or spasm and then we are guided somewhat as to whether we shall recommend surgery or not. If the case is decided to be stenosis we must even further carefully consider it, for as Dr. Porter brought out, some of the stenosis cases get well without operation. Yet the percentage is so small in consideration with the percentage of cases that die that it is not justifiable to wait and take that chance if the diagnosis of stenosis has been made. The other factor to be considered in these cases is the surgeon, the qualifications of the man who is going to do the work and what he is going to do. I do not believe that these cases should be operated upon indiscriminately, my fear is that the reports to-night will cause a good many babies' abdomens to be opened by incompetent men. It is not at all an operation to be rushed into. In the second place, what is the surgeon going to do when he does operate? It has only lately been pretty well decided that the gastroenterostomy offers the best chances. I might advise operation for gastroenterostomy where I would not advise operation for divulsion. If the operation is done by the best possible technic and by the best man, we have evidence that the results are good. All of the cases reported to-night have had good results, the only case of stenosis reported where the child died was the case allowed to go unoperated upon.

Dr. Barbat: There is not a great deal left to say upon this subject. The first case which attracted my attention was the child of a woman whom I delivered in 1904. The child did very well for two weeks, and then began to vomit, at first occasionally, then after every feeding. Change of diet made no difference, and a diagnosis of pyloric stenosis was made and operation advised. The parents changed doctors and the baby died at the age of six weeks. An autopsy revealed a stenosed pylorus, which would only admit a probe. The next case is the one which has been reported by Dr. Mohun. I confirmed the doctor's diagnosis, and agreed with him in regard to immediate operation, before the child became too weak. We operated on the 21st day after birth, and found on opening the abdomen at the site of the pylorus, a hard, glistening white mass the size of a marble. A no-loop posterior gastroenterostomy was done without reversing the jejunum, but keeping it in its normal direction. The baby was allowed some nourishment the same day, and at the end of five days was getting practically regular nursing. Although the operation is attended by many technical difficulties, the children take their anesthetic like milk and are ready for nursing in the afternoon. It is comparatively easy to do a gastroenterostomy on a dog or rabbit, or an adult, but when we attempt to operate upon a three weeks' old baby with a contracted intestine, it is a different matter. In this case the jejunum was no larger than an ordinary lead pencil, and it is not a particularly easy thing to place two rows of stitches between the stomach and jejunum. I have my doubts regarding the large number of cases reported as recovering under medical treatment alone, and believe that they are all to be classed under the spasmodic type and not the true stenotic type. During my trip east I inquired about this class of cases and received a good deal of information from the internes in some of the hospitals. I found that there have been a number of patients operated upon but not reported on account of the fatal results. There are a number of reasons for failure in these cases, faulty technic, and delay in bringing the patient to the surgeon being the principal ones. The medical man must bring the surgeon in consultation early, before the child has become too weak to stand operative measures. If this is done we will look for a very large percentage of recoveries in these otherwise hopeless cases. It is a mistaken idea that it is more dangerous to give a new born baby an anesthetic and operate upon it than to wait until it is older. I have done major operations on babies less than twenty-four hours old without any trouble, where it would probably have been fatal if I had waited.

Dr. Dudley Tait: My personal experience with the operative side of this question is limited to one case the clinical history of which was given to you to-night by Dr. Brown. In this case, after having failed to control the vomiting by means of a gastrojejunostomy, I made a gastroenterostomy on the fifth day but even then failed to get the desired result, the patient dying three days later without any local reaction. No autopsy having been performed nothing of interest can be elicited by further reference to this case. It has seemed to me to-night after listening to the numerous papers that this question has been viewed entirely from the weakest side of surgery, the mechanical side. After having perused the case histories in American and French literature no impartial observer will be convinced as to the cause of the congenital pyloric stenosis and therefore the propriety of surgical intervention in this condition. The question still remains unanswered, are we dealing with pure spasm or advanced pathological condition? Personally, I am inclined to favor the spasm theory, with gastritis as a possible factor, for the following reasons: first, the not infrequent onset of the clinical syndrome upon changing the infant's food; second, its variable date of occurrence often as late as three or four months after birth; third, the clinical and postmortem evidences of gastritis, mucus, pus and bacteria in the vomitus; fourth, the pathological findings in cases recovering under medical treatment and dying later from other causes, in one case as late as six months afterwards. In several such cases reported independently by Batten, Ibraham and Bloch, the autopsies showed hypertrophy and stenosis fully as marked as in the cases that had died after operation; fifth, the hypertrophy does not involve the stenotic ring alone but the entire region of the pylorus and generally the prepyloric area, the longitudinal and circular fibres being increased; sixth, in animal experimentation I have found that any injury involving the perpyloric area (elastic ligaments, etc.), will give a spasm of the pylorus, this spasm having been noted at autopsy as late as the ninth day. No sections, however, were made in this case and consequently I am not prepared to state whether or not any muscular hypertrophy existed; seventh, similar muscular hypertrophy has been found in other parts of the body; eighth, in the numerous reported cases of congenital pyloric stenosis in infants I can only find two in which tests were made to determine the presence of pyloric permeability; ninth, a very large proportion of cases recover under purely medical treatment. All these facts render it impossible to state definitely what part surgery is to play in the treatment of congenital pyloric stenosis in infants. When we remember that the Mayos are retracing their steps in gastric surgery, restricting the list of operable conditions, when we note that foreign surgeons who have had considerable experience with the operative treatment of infantile pyloric stenosis are becoming more conservative, we must admit that the surgeon must possess something more than mere mechanical views if he decides to invade what seems to be the medical man's domain.

Dr. MacMonagle: I have never had any cases of

congenital pyloric stenosis in infants. In a general way I am inclined to take the stand which Dr. Tait has taken. The question of the future health of the child operated upon and the action of the pyloric stenosis and the artificial opening after operation, seem to me very important and worthy of consideration. The reports of the future health of these cases will certainly be very interesting and instructive. In a number of cases of gastroenterostomy done upon adults, an autopsy some time after, has shown the pyloric stenosis cured and the artificial openings closed. In other cases, it has shown both the pyloric stenosis and the artificial opening closed. This condition of affairs is certainly very grave and should receive serious attention in coming to the decision of operating. Of course if there is an absolute obstruction of an organic nature, to the passage of material from the stomach to the intestines, there is only one thing to do in order to give the patient the slightest chance of relief, that is relieving the obstruction by making the junction of viscera in the way that seems best, or by removing the obstruction by leaving the pyloric stenosis as it is and opening a new channel from the stomach to the intestines. I do not mean this as a criticism upon the cases that have here been referred to to-night, as I believe these cases were carefully diagnosed and well treated. I merely want to raise the question of the future action, of the difficulties in such cases and in regard to the surgical procedure in all cases, as I fear that some enthusiasts may be led to surgical means as a relief before it is clearly established that a good result cannot be obtained by medical means.

THE FAUCIAL TONSILS CONSIDERED FROM A MEDICAL AND SURGICAL STANDPOINT.

By LOUIS C. DEANE, M. D., San Francisco.

The mere mention of the subject of this paper will bring to our minds thoughts, and probably experiences, we have had concerning these masses of lymphoid tissue.

Treat the subject as lightly as you will, nevertheless the prominent position of the faucial tonsils at the orifice of the respiratory and alimentary tracts, their exposed position to foreign substances, their close relation to the cervical lymphatics and their great vascularity gives them an importance not easily ignored. One remarkable feature is that these organs so ready of access and so easily observed have remained a kind of medical enigma.

What passes in the mind of the average observer when examining the tonsils? He notes if they are red, swollen and for the presence of exudate. If this trio are absent his investigation, as a rule, ceases and so the tonsils have remained for many years comparatively unmolested except for the tonsillotome (which is capable of removing a piece of the tonsil) and a long list of gargles and swabs.

Gradually the importance of a more serious consideration of the faucial tonsils has forced itself upon us. Aside from their local effect, their relation to certain systemic conditions has proven so intimate that in many instances our previous lethargy has changed to alarm.

Considering the faucial tonsils as a pathological entity they may be regarded from two points of view. First, diseases characteristic of themselves with their local effect and second as a portal of infection whereby the general economy may suffer from some apparently remote disorder.

By a gradual pathological chain the acute forms of tonsillitis can be merged into the chronic and so, various types of tonsillitis can, for clinical purposes, be described as a continuation of the same inflammatory disease. An acute superficial or catarrhal inflammation may readily subside or it may extend to the more severe form of lacuna tonsillitis, more commonly known as follicular, where the infection extends into the tonsillar crypts which accumulate a debris of epithelial cells, leukocytes and bacteria. These crypts form a favorable spot for the encouragement of bacterial growth and the tonsil reacts against it by inflammatory reaction. Let this condition carry us to the next type, i. e., parenchymatous inflammation where the stroma of the tonsil becomes involved; this may assume an acute condition where pus is formed from an infection of the surroundig tissue, as in peritonsillar abscess or quinsy. A more chronic course may be followed which resolves itself into organized inflammatory exudate in the form of scar tissue and an hypertrophy of the connective tissue. The acute lacuna tonsillitis just mentioned may assume, like unto itself, a chronic form the so-called caseous tonsillitis which consists of masses of inspissated secretion and bacteria mixed with food.

What the agent is that prompts these various changes and decides whether an acute superficial inflammation will subside as such or go on to graver forms can only be determined by knowing the nature of the infection which originally involved it in disease or attacked it later.

During the acute and chronic forms of tonsillitis many investigators have demonstrated the presence of pathogenic micro-organisms such as streptococcus, staphylococcus (aureous and albus) and diplococcus. In lesser frequency are found the pneumococcus, Kloebs-Laeffler bacillus, staphylococcus citreous, micrococcus tetragynous, micrococcus albus liquefaciens, bacillus tuberculosis, leptothrix and other forms.

Whether an acute attack can leave a permanent stigma upon the tonsil or whether it requires a long series of acute infections to accomplish the same end is merely a matter of degree; suffice it to say that the ill effect of the presence in the throat of an obstruction that can so impair functions and development, is a matter of serious consequence.

The effect of enlarged faucial tonsils upon articulation and deglutition is most marked. They can change the development of the mouth and nares such as faulty dental alignment, narrow palate arch and drooping lower maxilla. Note the dull and listless manner with stupid expression and thick lips. The local effect upon the mucous membrane of the entire respiratory tract from constantly breathing through the mouth causes a hacky cough and tendency to chronic bronchial affections. Their influence upon taste, hearing and smell is marked. Disturbed sleep with efforts at breathing has an influence upon the development of the chest and we see in these cases the so-called pigeon-breast.